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## Toxoplasmosis

BY
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**Department of Bacteriology, Sheffield University**

A PROTOZOAN parasite, *Toxoplasma gondii*, first isolated from a North African rodent, the gondi, by Nicolle & Manceaux in 1908, is the infective causal agent of toxoplasmosis. It is a strict parasite and, so far as is known, it multiplies only by longitudinal binary fission inside living animal host cells. A mature proliferative form is elongated lemon shaped, often flattened on one side, and is approximately 5$\mu$ by 1.5 to 2$\mu$ (Fig. 1). To reproduce, it burrows its way into a host cell, usually a reticulo-endothelial cell, and there divides repeatedly until 8 or 16 daughter parasites are formed (Fig. 2). By this time the host cell is exhausted if not dead, and the organisms contained within it are then dispersed by rupture of the cell membrane. The newly-liberated parasites tend to be thinner and even-shaped like a section of an orange because they have been packed together in the host cell with their long axes parallel. They enlarge in the host's extra-cellular tissue fluids, parasitise fresh cells and the cycle recommences. Eventually the host animal either dies from an acute generalised infection, or a balance is established as a result of the development of immunity by the host. We have little evidence that infection is completely eliminated in animals which recover. On the contrary, there is quite a lot of evidence to show that infection persists, but the parasites assume a different form within a cyst which has a well-developed membrane around it (Fig. 3). Subsequently, when and if conditions for growth become more favourable, either by a lessening of the host's immunity or by transfer to a non-immune host, the cyst ruptures and the contained forms grow, parasitise a fresh cell, multiply therein, and eventually cause another acute infection.

During the 16 years which followed Nicolle & Manceaux's discovery, members of most species of warm-blooded animals, including birds, were found to harbour this parasite. The evidence that cold-blooded animals can do so is not very convincing.

It was not until 1924, however, that the first evidence of human infection was obtained. Janku, a Prague ophthalmologist, found a cyst-like structure in sections of an eye from a patient with choroido-retinitis. At a later date this was identified as a Toxoplasma cyst. In 1939, Wolf, Cowen & Paige isolated toxoplasms from the brain of a child who died soon after birth. They did this by inoculating suspensions of brain material into mice. In 1941, Sabin demonstrated that the causal agent of meningo-encephalitis in two schoolchildren was toxoplasma, and a year later, in co-operation with Ruchman (Sabin & Ruchman, 1942) he showed that the presence of antibodies in the host could be proved by a neutralisation test performed in the skin of rabbits. Later in the same year, with Warren (Sabin & Warren, 1942), he developed a complement-fixing antibody test and in 1948, with Feldman (Sabin & Feldman, 1948), he published the " dye test " or " cytoplasm modifying test." These findings gave a great impetus to work on toxoplasmosis and, since then, most of the observations have been on man.

In humans, Toxoplasma can produce disease either before or after birth. In the former, a non-immune pregnant woman acquires a mild infection, and while she herself is rarely ill, the infection is passed on transplacentally to her foetus. The child may be born either dead or alive with internal hydrocephalus, intra-cerebral calcifications, choroiditis, and possibly with other neurological manifestations such as fits, palsies, etc. In other cases a generalised infection may be present at birth or develop soon afterwards and is characterised by fever, rash, adenopathy, and hepato-splenomegaly; this stage is often followed by the same neurological manifestations as in the previous group, suggesting that infection occurred earlier in pregnancy in the former group than the latter. The changes in the latter group are not always gross; they may be confined to the eye, even a small portion of choroid only, and remain unsuspected for years. Cases of congenital toxoplasmosis have been described in nearly all the civilised countries of the world.

Manifestations of the acquired type in humans are meningo-encephalitis, pneumonia of an atypical kind,

an exanthem not unlike typhus, and a regional or generalised adenopathy resembling glandular fever.

A diagnosis on clinical grounds alone is rarely justified but should be confirmed by laboratory tests. All the available procedures are equally applicable to animals and man. The most convincing test of all is isolation of the parasite by inoculation of body fluids or tissue suspensions into mice. Probably the best course is to inoculate six mice, each by both the intracerebral route (0.02 ml.) and the intraperitoneal route (up to 1.0 ml.) The former route is more likely to give positive results with the less pathogenic strains while the latter is more effective when there are only scanty organisms in the inoculum. The recipient mice in a positive case may die in 10 to 14 days from either a generalised infection, in which case the organisms are demonstrable in the tissues and peritoneal exudate, or from a meningo-encephalitis, when the organisms can be detected in brain smears. Sometimes the mice survive indefinitely as carriers but, to try to exhalt the virulence of the parasite, subpassages should be made at intervals of four to six weeks from those mice which have developed serum antibodies.

There is an intradermal test using an extract of killed toxoplasms. In humans and animals who have immunity, a delayed reaction of the tuberculin type develops and is usually maximal in 48 hours. The size of erythema which develops varies from 5 to over 100 mm. in diameter and the amount of induration is roughly proportional. We have not yet seen necrosis. Unfortunately this test gives no indication of the recency of infection as the size of erythema is not related to serum antibody titres, and its main use now is in population surveys.

The serum antibody tests now in general use are the complement-fixation test and the dye test. In humans, the complement-fixing antibodies disappear within a year or two of infection but the dye test antibodies persist for many years and, even though the titre gradually falls with the passage of time, there is no evidence that it ever falls below the level of detection. From these two tests some idea can be gained of the recency and grade of an infection.

To ascertain the diagnostic significance of antibody titres it was necessary to find out what levels were found in normal people. Many surveys have been done in several parts of the world and, as a result, many workers now regard a dye test titre in humans of 1:16 as suspicious, 1:32 as probable, and 1:64 or more as strong presumptive evidence of infection. Titres of less than 1:16 are thought most likely to be the results of remote mild infections. In an acute active infection in man, very high titres, in the hundreds or thousands, are obtained. A complement-fixing titre of 1:8 or more is regarded as evidence of active or recent infection. As with all serological tests, the demonstration of a rise in titre during the illness is of more value than a single high titre result. It seems likely that the titres developed differ in different animal species. Thus Erichsen & Harboe (1953) were unable to produce dye-test titres of over 1:40 in artificially inoculated chickens. We have found titres of 1:80,000 in rabbits, 1:400 to 1:800 in rats, and 1:100 in dogs when these animals have been artificially infected.

One of the problems still engaging a lot of attention is the sources of infection and the modes by which it is transmitted. In human congenital cases the child acquires its infection from the mother but we have no definite evidence as to where or how the mother derives hers. Neither, except in a few cases of laboratory infections, have we any knowledge of this in the acquired types. Only rarely has there been evidence suggesting man to man transmission. The frequency with which both husband and wife are positive is no more than is dictated by the laws of chance. On the other hand, many human cases have had an association with a sick animal, and on numerous occasions it has been possible to show that the animal had toxoplasmosis. Even more frequently, however, no history of close animal contact is obtained.

Further evidence in support of an animal source of human toxoplasmosis is the higher incidence of Toxoplasma antibodies in veterinary surgeons and abattoir workers and, in some areas at least, their greater frequency in country dwellers than in town dwellers.

This has led medical men to become more interested than usual in the doings of their veterinary colleagues. Five species of non-human primates—dogs, cats, ferrets, and silver foxes of the carnivores; the gondi, rabbits, hares, rats, mice, squirrels, guinea-pigs, and marmots of the rodents; insectivores; marsupials; pigs, sheep, and cattle of the ungulates; and hens, ducks, pigeons, canaries, and many wild birds have all been shown to harbour this parasite. I use the word " harbour " purposely because only rarely has it been shown to be pathogenic and then in peculiar circumstances to which I will refer later. Most frequently its presence was discovered as a result of surveys on apparently healthy animals.

### Dogs

There is no doubt that under some circumstances *T. gondii* can cause a severe disease in dogs. In 1910 Mello described a dog which had fever, anaemia, respiratory distress, and bloody diarrhoea. There was abdominal tenderness. After several days the animal died and at autopsy a sero-sanguineous exudate was found in the body cavities, small nodules in the lungs, and numerous small ulcers in the intestinal mucosa. Intra- and extra-cellular parasites were seen in films and sections. Since then, there have been over 60 cases described from all continents, and the clinico-pathological pattern has varied a great deal (Habegger, 1953). There is no sex predisposition, and pet dogs appear to be just as liable as strays. The onset of illness is usually insidious with fever, lassitude, anorexia, and diarrhoea, but sometimes it is sudden with vomiting followed by fits, paralysis, or other neurological manifestation. Rhinorrhoea and lachrymation are sometimes marked, and in other cases dyspnoea and pneumonia have been prominent. In roughly half of the cases the respiratory symptoms

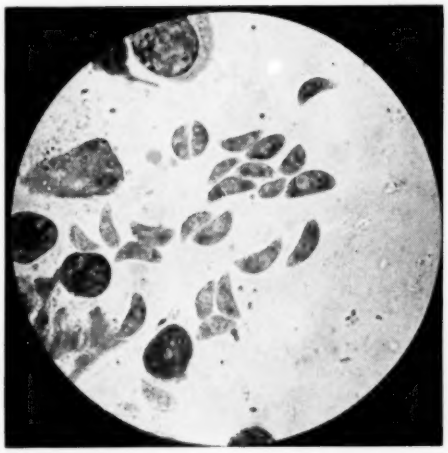

FIG. 1.—Extracellular *T. gondii* in mouse peritoneal exudate.

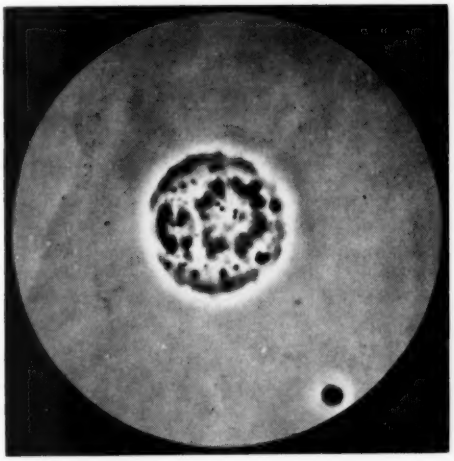

FIG. 2.—A cell which has been parasitised by *T. gondii* on two separate occasions. The earlier parasite has undergone several divisions while the later one has divided once only.

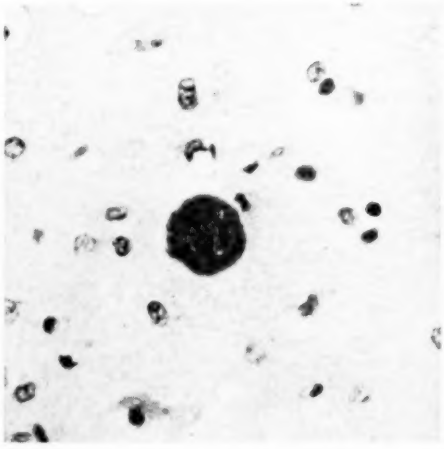

FIG. 3.—A cyst of *T. gondii* in a section of a human brain. The contained parasites are closely packed together and much smaller than the free forms. There is no surrounding inflammatory reaction.

have predominated, in a quarter the alimentary, and in the other quarter the neurological ones. In the lungs, necrotic nodules may be found in the parenchyma and a serous pleural exudate may be present. The hilar glands are swollen and toxoplasms can be found quite easily in the cells lining the alveoli or in those of the hyperaemic tracheal or bronchial mucosa.

In the digestive tract there may be small, deep ulcers in the hyperaemic jejunal or rectal mucosa, and organisms may be found in the adjacent mucosa or in the underlying musculature. Viable organisms have been obtained from the faeces. The liver and spleen are enlarged, the former often being lighter in colour than normal and having many miliary foci of necrosis. Here again toxoplasms are easily found in the liver cells, the biliary tract epithelium and the reticulo-endothelial cells of the spleen. The kidneys rarely show macroscopic lesions but organisms may be found in stained sections and in the urine. The heart muscle fibres may contain collections of parasites and there may even be foci of calcification. Blood and bone marrow may also contain them.

As far as the central nervous system is concerned, a lepto-meningitis may be present and foci or necrosis in the grey matter just beneath the ependyma. If the latter extend through the ependyma, portions of necrotic tissue may be shed into the ventricles and lead to an obstructive internal hydrocephalus. In the brain, organisms are rarely found in the free proliferative form; they are usually in cysts.

In differential diagnosis one must think of distemper in puppies and in older animals of leptospirosis, sinusitis, pneumonia, and gastro-enteritis. Histologically, one must differentiate Toxoplasms from Sarcosporidia whose cysts are much larger and have a very much thicker wall, from trypanosomes which have a kinetoplast, from encephalitozoon which stains well with Giemsa, and from Piroplasma where some of the forms are contained in red cells. Campbell (1956) rightly stresses the superiority of stained films over sections, and the need to examine several portions from each of a wide range of tissues.

One of the very surprising things about clinical canine toxoplasmosis is its apparent rarity and the great difficulty in producing it experimentally. We have given as many as 30,000,000 virulent R.H. strain organisms intravenously to a dog and it never developed any symptoms. Other people have had similar experiences. Jacobs (Jacobs *et al.*, 1955) was unable to produce clinical disease by feeding toxoplasms to dogs of any age, and even if they were given intravenously only those under 45 days of age developed signs of illness and died, while those older did not even become ill. Chamberlain and his co-workers (Chamberlain *et al.*, 1953) inoculated pregnant bitches intravenously with huge numbers of different strains of Toxoplasma and caused abortion or premature labour. Most of the offspring were infected and were born dead, or died of a generalised infection in the first few weeks. The milk of all the bitches contained virulent organisms. Some of them were quite well throughout, others were sick and recovered while others died. Olafson & Monlux (1942) have described the only experiment so far where there has been an apparent direct transmission from one sick puppy to its cage mates.

However, it is hardly likely that dogs will ever be naturally infected with such huge doses, and the probability is that most canine toxoplasmosis is subclinical and in this respect compares with that found in man. What could cause an ordinary subclinical infection to assume clinical severity? Sjolte (1948) thought that a prolonged period of cold weather was related to his finding toxoplasms in a Danish dog suffering from pneumonia. The same could be said of MacHattie's (1938) two cases of spontaneous toxoplasmosis in dogs in Baghdad during the month of March after a heavy snowfall in February. Campbell (Campbell *et al.*, 1955; Campbell, 1956) and his colleagues found evidence of toxoplasmosis in 18 dogs, all of which had clinical and histological evidence of virus infection. What part the Toxoplasma infection played in their illness is difficult to assess, for in seven of them it was generalised while in the other 11 it was sparse and sometimes confined to one organ, but it has been thought in other cases that concomitant infections played a part in activating Toxoplasma infection. Jacobs *et al.* (1955) found that feeding hookworm larvae in addition to Toxoplasma predisposed to active clinical toxoplasmosis. In humans, cases of concomitant tuberculosis and of bartonellosis have been described, and we had a case where Brill's disease (recrudescent typhus) was possibly associated with active toxoplasmosis. In dogs, youth and pregnancy have each been found to predispose to clinical infection, while in humans we have noted an acute exacerbation of choroido-retinitis in a woman who was pregnant and have found that most of the acquired human glandular type of toxoplasmosis is in children or young adults. Nicolle & Manceux's (1908) first findings were on gondii which had been kept captive and Rodhain's findings (1948) were on captive squirrels and a captive marmot. Cold, captivity, growth, pregnancy, and concomitant infections could all be called stress factors capable of upsetting adrenal function and its part in resistance to infection and the development of immunity.

### Cats

Only few observations have been made on cats. Otten (Otten *et al.*, 1951) and his co-workers tested the sera of cats and dogs in Hamburg and found all the cats negative while 49 of 122 dogs were positive. One of the cats tested was from the same house as one of the very strongly positive dogs. Wickham & Carne (1950) found pseudocysts in the brain of a cat dying from paralysis. No bacterial infection was found at autopsy even though all the viscera were hyperaemic. There was no inflammatory reaction around the pseudocysts and probably they were the result of a chronic infection unconnected with the death. They tried to infect kittens by feeding them with fresh infected rabbit kidneys but not only failed to produce disease but even subclinical infection. Olafson & Monlux (1942) describe a case of a one-year-old cat with fever, dyspnoea, anorexia, blindness, and gross enlargement of the abdominal glands.

At autopsy, foci of necrosis were found in the markedly enlarged mesenteric lymph nodes, and small ulcers with raised edges in the intestines, particularly the small intestine. In the lungs were white nodules up to 1 cm. in diameter. Isolated and aggregated toxoplasms were found in sections of the lymph nodes. Verlinde and Makstenieks (1950) found pseudocysts in brain sections of a six-year-old cat which died with signs of encephalomyelitis. On testing sera from the family which owned the cat they found they all contained Toxoplasma antibodies. They also obtained a history that an eight-year-old child had died six years before in the same house and that the child had a large head and blindness. This may have been a congenital toxoplasmosis but it would have been infected eight years before the cat was born and so the only connexion between the two episodes is the possibility of a persistent local source of infection.

### Rats

Perrin (Perrin *et al.*, 1943) found that 8.7 per cent. of Savannah rats were carriers of Toxoplasma cysts in the brain, and Eyles (1952) found 3.2 per cent. of Memphis rats to be carriers. Others in other countries have confirmed this work, but there are no records of any spontaneous disease in rats. This may be related to their resistance to huge doses given intraperitoneally in the laboratory. Even pregnant rats are unharmed but some of their foetuses are killed and resorbed while others, born alive, have mild cataracts (Giroud *et al.*, 1954).

### Rabbits

In 1909 Splendore described a rabbit which died in a wasted condition, having had paralysis and skin lesions. At autopsy, organisms corresponding with *T. gondii* were found. Since that time many observations on rabbit toxoplasmosis have been made, including epizootics in the Congo (van Saceghem, 1916), the East Indies (Brug *et al.*, 1925), France and Scandinavia. In the French epizootic there was a mortality of 30 per cent. (Marotel & Peirron, 1943).

We (Beverley *et al.*, 1954) found that 34 per cent. of wild rabbits in this country had serological evidence of having had Toxoplasma infection. In common with others we have found that 5 to 7 per cent. of tame rabbits do not develop clinical toxoplasmosis after injection with virulent Toxoplasma, and that a similar fraction have complement-fixing antibodies. The assumption is that they are immune because they have already had a subclinical infection. Lainson (1955) has shown that such rabbits carry toxoplasms in their brains and we have been able to confirm his finding. We have found them in rabbits which were bred in our breeding station and which had never been ill. Two strains were recovered from such rabbits and both of them failed to produce disease not only in rabbits but also in mice which are usually regarded as highly susceptible. The virulence of one of these strains was exalted by inoculating it into mice treated for seven days beforehand and seven days subsequently with cortisone. This exalted strain now kills mice regularly in five to six days and rabbits in a week, while the original strains are still avirulent.

### Hares

In Bornholm, Christiansen (1948) found evidence that Toxoplasma caused the death of 30 per cent. of hares which he autopsied. In Denmark, Christiansen & Siim (1951) discovered outbreaks of clinical toxoplasmosis in hares during the winter months. They found very little evidence of mild or subclinical infection. Nearly all the cases were acute, fatal, general systemic infections. The spleens were hyperaemic and markedly enlarged. The livers were enlarged, light coloured, and degenerate with scattered submiliary necrotic red-yellow foci sometimes surrounded by a narrow haemorrhagic zone, and frequently they contained scattered haemorrhages; the mesenteric lymph nodes were swollen hard and injected, and sometimes contained haemorrhagic necrotic foci. The lungs were distended, oedematous, and hyperaemic and sero-sanguineous fluid was present in the pleural cavities. The general nutrition was good, indicating that the disease had run an acute course. Histological examination showed toxoplasms in large numbers in most organs, with pathological changes in the lungs, liver, spleen, and, to a lesser extent, in the brain.

### Birds

Toxoplasma infection has been described in about 45 species of birds but many of these claims were based on histological appearances alone and so are not wholly acceptable. Two species in particular may be mentioned. Jacobs (Jacobs *et al.*, 1952) found evidence of an infection rate of 12.5 per cent. among pigeons caught in the precincts of the Capitol in Washington. The strains isolated were in every way comparable with the virulent R.H. strain which was originally isolated from a human. He found that after artificial infection half of the pigeons remained well but the parasites were present in the blood in high concentration for as long as six weeks. He thought pigeons might be a reservoir from which transmission to other species could occur. Erichsen & Harboe (1953) have described an outbreak of a fatal disease in White Leghorn hens on a farm in S.E. Norway. (The fowls on neighbouring farms were unaffected.) Some of the fowls were found dead without having shown any previous signs of illness while others were visibly ill for up to a month before they died. Signs of illness were anorexia, emaciation, pallor, and shrinking of the comb. Some of the fowls had diarrhoea and others seemed to be blind because they might walk into walls, people, or animals. Histologically, toxoplasms were found in various organs and the accompanying lesions were pericarditis, focal or diffuse myocarditis, focal encephalitis, necrotising hepatitis, and ulcerating gastro-enteritis. In three, diffuse choroiditis was found but no toxoplasms were seen. A surprising finding in both the pigeons and the fowls was that some of the birds from which toxoplasms were isolated had no serum antibodies.

### Cattle

Chamberlain & Cole (1953) and their co-workers described an outbreak of fatal illness in a herd of Brown Swiss cows. 45 of 78 calves, born over a period of a year, died at ages ranging from one day to six months. There had been no abortions. The majority developed dyspnoea, rapid respirations, coughing, sneezing, nasal discharge, frothing at the mouth, trembling and shaking of the head, and dehydration. Temperatures as high as 106° F. were recorded, but many were normal or even subnormal. Finally, weakness, grinding of the teeth, and prostration developed, followed by death in two to six days. Some died suddenly without exhibiting previous illness while in others the course of the disease lasted six months. Diarrhoea was not a constant feature, but when it did occur it was accompanied by blood and mucus. Some died after showing only extreme depression while others gradually recovered.

One four-week-old calf which had been sick for two days was examined physically and pathologically. In addition to many of the signs already mentioned, it had tenderness on abdominal palpation, soreness of the leg joints, and excessive fluid in the carpal joints. Brucella agglutination tests were negative. At autopsy, fibrinous deposits were found in the peritoneal cavity, the submaxillary and bronchial lymph nodes were swollen, oedematous, and haemorrhagic, the trachea contained mucofibrinous masses and transudate, and pneumonic consolidation was present in the lungs. Clear fluid was present in the pericardium but the C.S.F. was cloudy and pink. A strain of Toxoplasma was recovered by inoculation of suspensions of lung tissue into mice, and toxoplasms were found histologically in lesions of the brain, lung, and lymph nodes. There was no evidence of bovine encephalomyelitis. These workers also found sporadic cases in three other herds and were able to recover toxoplasms from the colostrum of one cow.

### Swine

Farrell, Docton, Chamberlain & Cole (1952) showed that toxoplasms were responsible for an outbreak of infection in a herd of swine in Ohio. There had been a recurring, undiagnosed disease for many years which had assumed serious proportions over the previous two years. In their investigation 11 sick hogs ranging from one day to over a year old were examined. They succeeded in isolating toxoplasms from two of them and they found that healthy hogs, purchased from a disease-free farm, became sick when placed on the infected farm. Weinman & Chandler (1954) confirm the natural occurrence of toxoplasmosis in pigs, but in a subclinical form, not as an illness. They found swine remarkably resistant to experimental infections with huge doses of Toxoplasma.

### Discussion

Enough has been said to show that toxoplasmosis can cause local outbreaks of disease in animals. So far, the only evidence of widespread epizootics of clinical illness has been among hares in Sweden and Denmark. The impression is that it is much more usual for toxoplasms to produce only mild or subclinical infection.

We know that toxoplasms can be found in the blood, faeces, urine, respiratory secretions, and tissues of many species of animals. Possible modes of transmission are contamination of foodstuffs by faecal or urinary excreta, the intermediary of blood-sucking insects, ingestion of cooked infected meats, and inhalation of infected aerial suspensions. Numerous experiments using many different kinds of insect vectors have failed to transmit infection from an animal with a known parasitaemia to others of the same species. We cannot rule out this method but the accumulation of negative evidence so far does suggest that it is not very likely. Cannibalism and flesh eating are certainly possibilities and the great natural resistance of carnivores to experimental infection is of interest in this connexion. Mice will readily develop a fatal generalised infection if they are allowed to eat infected tissues but not if they ingest liquid exudate rich in toxoplasms. This suggests that in mice the gastric fluids quickly kill readily accessible forms but not those inside pieces of tissue. Hydrochloric acid at concentrations equivalent to those in gastric juice will kill free toxoplasms in 15 minutes. One can easily understand how dogs eating rats, and cats eating rats or mice, and carrion birds eating carcases of dead animals can become infected in this way, but humans and herbivores like rabbits do not qualify as candidates for infection by this means. Faecal or urinary contamination of foodstuffs could cause infections in these animals, but it must be remembered that toxoplasms in the proliferative phase are not very resistant to cold and drying. The cyst form is likely to be more resistant. With regard to the respiratory route there are a few observations worthy of mention. It is far easier to infect mice and rabbits by the nasal route than by the alimentary one; in humans with the glandular type of illness, the glands most frequently involved are the cervical ones; Cathie (1954) has shown that toxoplasms were present in the saliva of a boy with glandular toxoplasmosis, and Pinkerton & Henderson (1941) have found them in the bronchial secretions of a patient who died from a pneumonia-like illness.

In conclusion I would like to say something about treatment. Experimental infections can be successfully treated by chemotherapy. The most successful method so far is a combination of sulphamezathine and daraprim. Sulphamezathine alone will tide most animals over the acute stages of the disease but the addition of daraprim goes a long way to prevent either relapses or the gradual passage of an acute infection into a chronic one or the carrier state. In certain circumstances recrudescences occur and the pathology is then further complicated by the presence of a hypersensitive state. Cortisone is a useful adjunct to chemotherapy in these cases.

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(*Concluded at foot of page* 342)

# Endocrine Alopecia in the Dog

BY

**E. A. CAMPBELL and P. H. MORTIMER**

**Department of Veterinary Clinical Studies, Cambridge**

COFFIN & Munson (1953) have recorded alopecia of endocrine origin in the dog. Certain of their cases were classified as "Canine Cushing Syndrome," presumably on the size and weight of the adrenal at *post mortem*, since blood-sugar values were normal and hypogonadism was present in some cases. The following case appears clinically identical with those of Coffin, but despite detailed investigation we have been unable to obtain evidence of hyperfunction of the adrenals or pituitary.

## History

The subject was a nine-year-old Bedlington Terrier dog weighing 41 lbs.

Clinical signs were first seen some four to five months prior to examination. The dog commenced to gain weight which showed mainly as an increase in size of abdomen. Excessive thirst and lassitude were observed, and urinary incontinence had caused the owners nocturnal disturbance. The hair loss was first seen on the limbs and over the shoulders and bilateral extension of these areas along the sides of the thorax and abdomen became obvious. Hyperkeratinisation of all pads developed contemporaneously with the hair loss. An adequate course of thyroid treatment had failed to ameliorate the condition.

## Clinical Examination

### *General*

The dog stood in a rather straight-limbed posture, this being more obvious in the hind quarters. The abdomen was enlarged but by no means obese, and the limbs did not share the weight increase which had occurred. The nipples and penis appeared normal, the testes were equal in size but seemed smaller and softer than is usual. On opening the mouth, diffuse pigmentation was noticed on the upper lips and gums.

### *Hair*

The marked alopecia was present, in greater or lesser degree, on most parts of the integument, except the head which had normal hair covering. Along the neck and dorsal part of the thorax and lumbo-sacral regions, the hair was very fine and down-like in texture. This "down" was also present on all four legs below the carpus and hocks (Fig. 1).

### *Skin*

A generalised diffuse pigmentation was noticeable on the hairless parts, there was no erythema or scaling and no pruritis. More discrete areas of heavy pigmentation (macules) were to be seen on both sides of the body below the mid-line, and also on all four

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legs down to the hocks. The pads on all four feet and the stopper pads were grossly hyperkeratinised, and so exaggerated that the tubes of keratin were overgrown and turned outwards, so projecting from the sides of the feet and appearing rather like large roughened supernumerary claws. The claws themselves were consequently overgrown but normal in structure (Fig. 2). There was only mild hyperkeratosis on the nose.

FIG. 1.

FIG. 2.

Haemoglobin was 18.6 grammes per 100 ml., W.B.C. 14,000 per c.mm., eosinophils nil; basophils nil; lymphocytes 9 per cent.; monocytes 7.5 per cent.

X-ray examination showed a normal skeleton with no evidence of osteoporosis.

The reported polyurea appeared exaggerated, the 24-hour urinary volume averaging 500 ml., a value only slightly in excess of that found here for normal dogs allowed fluids (especially milk) *ad lib*. *Diabetes insipidus* in the dog has been reported by Sanders, Stephenson & McEntee (1951), and they cite references giving a urine volume of 20 litres *per diem*.

GLUCOSE AND INSULIN SENSITIVITY TESTS

| | |
|---|---|
| *Blood Analysis* | |
| Total serum protein ... | 5·3 mg. per cent. |
| Plasma Fractionation : | |
| Albumen ... ... | 44·8 per cent. |
| Globulin $\alpha$ ... ... | 24·8 „ |
| „ $\beta$ ... ... | 16·2 „ |
| „ $\gamma$ ... ... | 14·4 „ |
| Blood : | |
| Urea ... ... ... | 97 mg. per cent. |
| Inorganic $PO_4$ ... | 2·2 „ „ |
| Calcium ... ... | 10·8 „ „ |
| Serum Na ... ... | 133 meq. per litre |
| „ K ... ... | 6·9 „ „ |
| Blood sugar ... ... | 52 mg. per cent. |
| Urinary 17-ketosteroids | 0 to 0·23 mg. per 24 hours |
| 24-hour urine volume... | 480 to 540 c.c. (mean 500) |
| S.G. ... ... ... | Mean 1,010 |
| Albumen present in urine. | |

In pituitary basophilism the retention of sodium and loss of potassium are well-established facts and usually accompanied by hyperglycaemia and glycosuria. X-ray examination shows marked osteoporosis (Albright & Reifenstein, 1951), and polycythemia is generally present.

None of these signs were found, but in view of the blood sugar and Na and K figures it was decided to investigate pituitary and adrenal cortical function employing glucose tolerance and insulin sensitivity tests.

Glucose was given *per os* at the level of 1 gramme per lb. bodyweight. As insulin sensitivity tests have not been reported in clinical investigations in the dog, recourse had to be taken to human methods (Fraser, Albright & Smith, 1941), and experimental work on hypophysectomised dogs (Chaikoff, Reichert, Larson, Mathes, 1935). Chaikoff *et al.* used a dosage of 1/16 unit of insulin per kilo bodyweight, Fraser *et al.* 1/10 unit. In view of the slight hypoglycaemia found it was deemed advisable to employ half this dosage, *i.e.* 0.05 unit per kilo. Results are shown in Fig. 3 (overleaf).

## Discussion

De Bodo, Block & Grass (1942) give blood sugar for hypophysectomised dogs as 33 to 69 mg. per cent. with a mean of 53 mg. per cent.; further these dogs were 30 to 60 times as sensitive to insulin as normal animals. Chaikoff *et al.* (1935) found that the blood sugar of normal dogs given 1/16 unit of insulin per kilo bodyweight fell 5 to 8 per cent. while hypo-

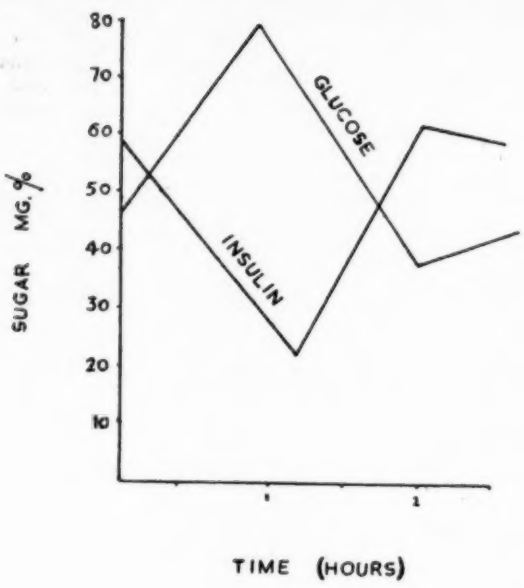


FIG. 3.—Glucose Tolerance and Insulin Sensitivity Tests.

physectomised animals had changes of 21 to 52 per cent., consistent with the results here. These same dogs showed a lowering of 16 to 41 per cent. in serum phosphate. These findings occurred when secondary changes to hypopituitarism had become established.

Although the low 17 ketosteroid value for a male is suggestive of pituitary hypofunction, this assumption would not appear justified in the dog without further supporting evidence. Paschkis *et al.* (1943) reported total urinary 17 ketosteroids in the dog to be 0.5 to 1.5 mg. per 24 hours, irrespective of sex, suggesting testicular androgens in the canine undergo a different form of metabolism, or are produced in small amount.

The same workers were able to show that testosterone fed to the male dog did not cause a rise in 17 ketosteroids which are presumably representative in this species of the "N" hormone of Albright (1942). While the blood electrolyte figures, in the present case and those of Coffin & Munson suggest hyperkalaemia, support for malfunction of the non-oxygenated $C_{11}$ steroids is weakened by the presence of kidney malfunction.

The level of blood urea is in agreement with experimental work on cortical hypofunction (Swingle *et al.*, 1934), but this and the lowered plasma albumen, while symptomatic of adrenal hypofunction could be the result of kidney damage.

Coffin & Munson have pointed out that the areas of hair loss are related to the sites of wear and contact, although the primary disturbance is of endocrine origin. It may well be that our case (although clinically identical with theirs) arises from a different primary cause. It would seem to us that in both instances the findings resemble those of the third stage of the Adaption Syndrome (Seyle, 1949).

**Summary**

A case of alopecia in the dog is described. Similar cases previously reported have been claimed to resemble Cushing's syndrome in man. Further metabolic investigation in the case presented would not appear to substantiate the claim of pituitary/adrenal hyperfunction.

*Acknowledgments.*—We are indebted to Mr. T. F. Tunney of Peterborough for presentation of this case.

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**Addendum**

Subsequent to the submission of this paper the animal was destroyed. The owners in granting permission for *post mortem* stipulated that euthanasia be performed by their own veterinary surgeon. Due to transport delays, *post-mortem* changes were too advanced for accurate histological examination by the time the dog was received.

The adrenals could not be identified until a diligent search had been made of the retroperitoneal mass. They were just under 2 centimetres in length and dark brown in colour. No clearly defined zones could be seen under the microscope. The pituitary was not enlarged. The kidneys were of normal size and showed no fibrosis or evidence of chronic nephritis. The thyroid lacunae contained much colloid suggesting hypofunction. The mucous membrane of the cheeks, gums, hard palate, and uvula showed patchy pigmentation similar to that seen on the skin. The first few feet of the duodenum were unique in displaying numerous raised circumscribed haemorrhagic areas about 5 to 7 mm. in diameter. None of these had ulcerated. These findings resemble those of adrenal cortical atrophy in man, and the only reported cases in the dog (Hadlow, 1953) found at autopsy. However, in view of the age of the animal, and as Addison's Disease has not been reported in the dog, we feel caution should be used in postulating that the pituitary adrenal hypofunction is the primary origin.

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# A Suspected Case of Congenital Porphyria (Pink Tooth) in a Heifer

BY

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## Introduction

CONGENITAL porphyria is a rare metabolic disorder of animals and man which is characterised by a high level of porphyrins in the blood, leading to their deposition in various tissues and organs of the body. The teeth are usually a browny-pink colour, the pigment being deposited mainly in the dentine. The bones are usually distinctly brown in colour, and on transverse section are seen to contain concentric rings of lighter and darker brown pigment. The urine is reddish-brown and the kidney is darker in colour than normal. Lymph glands, liver, lung, and other organs may also be discoloured. Cartilage, ligaments, and tendons are usually normal. The porphyrins are photosensitising agents and lesions due to photosensitivity are commonly seen on the skin after exposure to sunlight. Growth may be retarded and anaemia may be present.

Fourie (1936) recorded the occurrence of this condition in a herd of Shorthorn cattle in South Africa after the introduction of a pure-bred stock bull. The 13 recorded cases, approximately three-quarters of which were males, were all sired by this bull. As a result of breeding experiments (Fourie, 1939) he stated that the disease seemed to be inherited as a simple Mendelian recessive character. Rimington (1936) examined blood, faeces, urine, liver, kidneys, and other organs from affected animals and considered that the disease was probably caused by an abnormality in the synthesis of haemoglobin, due to the lack of a specific catalyst or enzyme, resulting in an accumulation in the body of porphyrins belonging mainly to the abnormal isomeric series I.

Clare & Stephens (1944) recorded congenital porphyria in pigs in New Zealand, and more recently Jorgensen & With (1955) have recorded this condition both in pigs and cattle in Denmark. A recent American publication (Fincher *et al.*, 1956) mentioned two affected Holstein calves from different farms in California which "are to be described in detail elsewhere."

There have been several reports of dark-coloured bones in abattoir material, summarised by Fourie (1936). Thornton (1952), in his *Textbook of Meat Inspection*, has described a condition seen in slaughter-houses in the carcases of calves, young cattle, and pigs in which all the bones of the skeleton are a dark brown chocolate colour, and in which the teeth, lungs, kidneys, liver, and lymph nodes may also be involved. It seems likely that this is, in fact, congenital porphyria.

Three live affected young cattle (Shorthorns) have recently been discovered on a farm in Buckinghamshire by Loosmore, and two of these have been demonstrated to the Physiological Society (Amoroso, Loosmore, Rimington & Tooth, 1956).

### *Subject*

A two-year-old light roan Shorthorn heifer.

### *Clinical History*

This animal was first turned out to grass as a yearling in May, 1955, and was one of a group of about half a dozen heifers. Some days later this animal was noticed to have lesions of photosensitisation along the centre of her back, and as the condition was becoming worse, she was returned to a loose-box. The lesions along the back subsided, and the heifer remained under cover until March, 1956, when she was again turned out to grass, and the lesions of photosensitisation reappeared. At this time the animal showed marked stunting in growth, and professional advice was now sought.

### *Clinical Examination*

Dry, scab-like areas, yellowish-grey in colour, were present along the heifer's back. The animal was in poor condition, and although she was almost two and a half years old, no permanent incisors were present. There was a striking, browny-pink coloration of the teeth, mainly in the dentine. The mucous membranes of the eyes and of the mouth were very pale and anaemic looking. In the course of examination an approximately normal volume of urine was passed by the heifer, and it was noticed that this was a reddish-brown colour, and was at the time assumed to be blood-stained.

No other abnormalities were detected, but as a good prognosis could not be given, slaughter was agreed upon.

### *Post-mortem Examination*

The main finding at *post-mortem* examination was that in addition to the discoloration of the teeth, the bones were brown in colour throughout their substance. There was very little urine in the bladder, and no abnormality of this organ or of the kidney could be seen to explain the reddish-brown urine, although the kidney was rather darker in colour than normal.

The teeth and bone were subsequently examined in ultra-violet light, when a bright red fluorescence was observed—a characteristic of porphyrins.

### *Herd History*

A return visit was made to the farm in August, 1956, in order to carry out further investigations.

It was established that the heifer which had been slaughtered had been bred on the farm. Her dam had had one calf before being bought, but nothing was known about this calf. The affected heifer was her second calf. She had subsequently had another heifer calf, which was a full sister of the affected animal and was still on the farm. This heifer was examined, but no abnormality in growth rate, teeth, or mucous membranes could be detected. Several other heifers and calves sired by the same bull were similarly examined, but again no abnormalities were detected.

Both parents of the affected heifer had been slaughtered some months previously—the bull because his daughters were ready for service, and the cow because her milk yield was not high. Nothing unusual had been noticed by the farmer in these animals during life, with the exception that the dam " got very hot and sweaty along the back in hot weather " and because of this she was always brought under cover.

### Discussion

Since in the present case only one affected animal has been seen, it is unwise to state categorically that this is a congenital condition. However, if, as Fourie has shown, the congenital form is a simple recessive, one would only expect to see symptoms of porphyria in one quarter of the offspring of parents heterozygous for this condition. In the case recorded, only two such offspring were seen, one of which was affected and the other was not.

An acute idiopathic (toxic) porphyria, which is inherited as a Mendelian dominant character, has been described in man (Waldenström, 1937), symptoms including dark-coloured urine, abdominal pain, vomiting, and colic. No pigment is deposited in bone or other tissues, and photosensitivity is absent. Waldenström also recognised *Porphyria cutanea tarda*, characterised by sensitivity to light and excess porphyrin excretion in the urine, with occasional colics, but no discoloration of the teeth as is seen in congenital porphyria. This disease also comes on later in life (usually about puberty or after), which is another feature distinguishing it from congenital porphyria. Symptomatic porphyria is also described as resulting from poisoning from a variety of substances, *e.g.* lead, sulphonal, barbiturates, but here there is no familial background, and again both photosensitivity and pigmentation of teeth and bones are absent.

In the present case the fact that there has been deposition of pigment throughout the bones and teeth suggests that the condition has been present since calfhood, and it seems reasonable to assume, therefore, that it is in this case congenital.

The present case showed symptoms very similar to those reported by Fourie in South Africa. The lesions of photosensitisation around the eyes and nose were not obvious, possibly because the farmer was keeping careful watch on this animal when she was turned out to pasture the second time, and as soon as lesions along the back were seen she was returned to her loose-box. The bones were an even dark brown rather than showing concentric rings of light and darker pigmentation. Discoloration of lung, liver, and lymph glands was not noticed, but may have been missed at *post-mortem* examination.

Jorgensen and With did not mention photosensitisation as a symptom in their cases in cattle in Denmark, but they were more concerned with the nature of the porphyrins in the body, rather than clinical symptoms. They did report the owner of one of the animals as having said that it got on much better indoors than at pasture. Neither they nor Clare and Stephens in New Zealand saw photosensitisation in affected pigs, and the health of these pigs was little, if at all affected.

It seems likely that congenital porphyria is more common than is generally supposed. Affected cattle, as in this case, are usually unthrifty and are probably slaughtered. Hence, as Thornton has reported, it is seen in the carcases of calves and young cattle. In pigs no abnormality is usually detected until discoloured bones are seen after slaughter.

### Summary

The occurrence of a suspected case of congenital porphyria in a Shorthorn heifer in Staffordshire is recorded. Both individual and familial histories are described.

*Acknowledgments.*—I should like to thank Mr. J. A. Lee, B.SC., M.R.C.V.S., of Uttoxeter, with whom I first saw this case, for permission to write this article. I am grateful to Mr. A. R. Jennings and Dr. J. Keilin of the School of Veterinary Medicine, Cambridge, for assistance in diagnosis and for providing some of the references, and to Professor C. Rimington, F.R.S., of University College Hospital, London, for his advice and encouragement.

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## HOLIDAY EXCHANGE

*France*

Sixteen-year-old French boy, whose family live in a villa with garden by the sea at Marseilles, asks to be put in touch with an English child for successive exchange visits of a month each in England from July 13th to August 13th, and in France from August 13th to September 13th, the children spending the whole time together and travelling under the care of L'Amitié Internationale des Jeunes (London office: 28, Duke Street, St. James's, S.W.1).

# Current Literature

## BOOK REVIEW

**Animal Nutrition.** By L. A. MAYNARD and J. K. LOOSLI. (4th edition.) 484 pp., 41 tables, 50 figs. McGraw-Hill Co., London. Price 56s. 6d.

In the different fields of knowledge there are text-books which go through one edition after another. Not seldom the original author gives place to a second and he in turn to a third, so that the life of the book may well span that of two or three men. One recalls, for example, what was for a long time " Halliburton's ' Physiology,' " though Halliburton was not the original author, as well as the text-book of physiology taken over from Starling by Lovatt-Evans.

It is of some importance to attempt to discover what qualities the original book must possess in order that a steady flow of editions may then follow. Doubtless the process is facilitated if the subject appeals to a large number of people, for then the price can be fixed at an attractive level. But most important of all it seems necessary that the primary author should have a strong sense of the way in which the subject is likely to develop. With such a guiding instinct the writer is likely to lay-out his subject matter in a way which will permit smooth development, with a minimum of disturbance of large portions of the text, in subsequent editions. Quite clearly there must normally be a substantial body of information available for initial classification; equally clearly, it is advantageous if the author is working actively in the selected field and if he possesses enthusiasm for his subject.

There are signs that Maynard's " Animal Nutrition " may be a book of the kind under discussion, for this 1956 edition is the fourth within the space of 20 years, whilst there have been so few changes in the general plan of the successive editions that in the present case it has been possible to insert a completely new, well-balanced chapter on antibiotics and related feeding additives with as much smoothness as if a place had initially been reserved for it. Moreover, the first edition appeared at a time when there was indeed a substantial body of information ready for classification, for the last section of nutrients to be discovered, namely the vitamins, was beginning to take on its final shape. Lastly, both Professor Maynard and the junior author who now joins him have long been engaged in nutritional research.

Every edition so far has been of roughly the same length (some 500 pp.), new material being added each time to take the place of " old wood." In their Preface to the fourth edition the authors make it clear that they aim primarily at American readers. This seems a right attitude to adopt, for anyone who strives too hard to catch the ear of the world may well find later that no one has been listening. Nevertheless, it has always been a book which may be read with profit by Europeans as well as Americans: in fact, in the field of basic animal nutrition it is probably the best text in the English language. Moreover, though intended for American use initially, it displays none of the myopia which now tends to spoil some American texts, as it once used to do German books, a myopia which renders invisible anything more remote from the U.S.A. than the nearest Atlantic or Pacific breaker. In this edition, for example, there are numerous references to European work of the last few years, whilst in the Preface the authors explain that on grounds of simple availability of journals, they have had to use a preponderance of U.S. references.

As previously the book is divided into four sections, dealing with the general bases of nutrition, nutrients and their metabolism, the measurement of body needs and feed values, and nutritive requirements for body processes and productive functions, respectively. Apart from the new chapter already mentioned, the major changes lie in the addition of fresh material dealing with recent discoveries relating to nutrients and to techniques. It is not to be expected that such additions will change drastically the face of a book so frequently revised: instead, one finds here and there new paragraphs dealing with a modern trend, *e.g.* " Indicators in pasture and range studies," or an extension of the subject matter devoted to vitamin $B_{12}$. A new departure is an Appendix containing the recommended nutrient allowances for live-stock as specified by the National Research Council of the U.S.A.

The printing, paper, and general production are excellent. Only one (?) printing error has been noticed, that of Hildith for Hilditch (p. 441), whilst the Subject Index seems quite competent. On the other hand it seems odd to omit from the Author Index the names which occur in the lists of " Selected Literature " following each chapter. It results in the inclusion of Arrhenius, a distinguished scientist but not a nutritionist, whereas Reyniers, a pioneer in nutrition studies with germ-free animals, is omitted.

Although this book has not been written primarily for veterinary surgeons, there are numerous references to disease of nutritional origin. It contains much material of interest both to students and practitioners, even though the formulation of rations is described only in general terms. Lastly, it is pleasant to observe that the growth of the subject, with the consequent need for pruning of the text, has not led to the elimination of the biographical footnotes. It may well be that a consciousness of the history of animal nutrition is not the least of the factors responsible for the logical development of the subject which this book continues to show.

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## B.O.C.M. POULTRY AWARD

The 1956 B.O.C.M. Poultry Award was presented to Dr. R. F. Gordon at a special luncheon held in London recently, and leading members of the veterinary profession, the Ministry of Agriculture, and the poultry industry were present.

# The R.C.V.S. Council Election

The election will shortly take place of five members of Council, R.C.V.S., as announced in our issue of March 2nd. The following gentlemen have offered themselves as candidates: Mr. L. G. Anderson, of Aylesbury, Mr. G. Atkinson, of Exeter, Professor R. Lovell, of London, Mr. T. M. Mitchell, of Stockfield, Dr. R. F. Montgomerie, of Beckenham, and Mr. T. Stewart, of Edinburgh. Brief biographical details of the candidates are as follows:—

**Mr. L. G. Anderson,** D.V.M., M.R.C.V.S., was born in 1909. He was educated at the Haberdashers' School, London, and attended a course in Agriculture at Reading University from 1928–9. In 1929 he emigrated to Canada, and after farming there for three years, he entered the Ontario State Veterinary College and graduated in 1936. He then returned to London and graduated from the Royal Veterinary College three months later. He took over his present practice in Aylesbury in 1937, and is now a partner in Anderson & Bugg.

He has been President of the Royal Counties Division of the B.V.A., was elected a Member of the N.V.M.A. Council in 1942, and was President of N.V.M.A. from 1947–8.

**Mr. G. Atkinson,** M.R.C.V.S., was born in South Shields in County Durham and was educated in Edinburgh and at the Royal Veterinary College, London, qualifying in 1914. After a few months in general practice he joined the Northumbrian Territorial Division as Veterinary Officer and went with them to France in the early part of 1915. He served in France, Mesopotamia and India (North-West Frontier).

Demobilised in November, 1919, he then joined the Ministry of Agriculture as Veterinary Inspector in February, 1920. Before being appointed to Exeter as Superintending Veterinary Officer in 1938, the principal places in which he was stationed were the Laboratory at Weybridge (four years), Bristol (four years), Head Office, London (four years) and Newcastle upon Tyne (three years). Mr. Atkinson was promoted to Divisional Inspector in 1925 while at the Laboratory, and to Superintending Veterinary Officer in 1938. He joined the B.V.A. in the early 1920s and has been a member of the Council for about seven years. He is chairman of the Veterinary State Medicine Committee; he is a member of the Mid-West and Western Counties Divisions of the B.V.A. and has been President of the latter. He is a member of the Rotary Club of Exeter and an associate member of the Royal Society of Health.

**Professor R. Lovell,** PH.D., D.SC., M.R.C.V.S., D.V.S.M., was born in 1897, and was educated at Hardye's School, Dorchester, and at the Royal Veterinary College, London, qualifying in 1923. He was awarded his D.V.S.M. (Manchester) in 1925, Ph.D. in Bacteriology (London) in 1932, and D.Sc. (Manchester) in 1946.

Professor Lovell was Demonstrator in Bacteriology in the Department of Bacteriology and Preventive Medicine of the University of Manchester 1925–7; Lecturer in the Division of Bacteriology and Immunology, the London School of Hygiene and Tropical Medicine, 1927–33; Head of Division of Bacteriology in the Research Institute in Animal Pathology, Royal Veterinary College, 1933–53, and became Professor of Bacteriology in the Department of Veterinary Pathology, University of London Veterinary School.

He was the Almroth Wright Lecturer for 1950, and has been awarded the Dalrymple-Champneys Cup and Medal. In 1955 he was Distinguished Visiting Lecturer to Michigan State University, and in 1957 the Benjamin Ward Richardson Lecturer. He has been President of the Comparative Medicine Section of the Royal Society of Medicine, and is now Hon. Treasurer of the Society for General Microbiology. He has visited East and West Africa to advise on Higher Education on behalf of the University of London.

**Mr. T. M. Mitchell,** M.R.C.V.S., D.V.S.M., was born in 1894. He was educated at the Sunderland Bede Collegiate School and the Royal (Dick) Veterinary College, graduating in 1914, and was awarded his D.V.S.M. in 1921.

He joined the R.(D.)C.V. O.T.C. in 1921 with special rank, was veterinary assistant in the Scottish Command for five months in 1914, and was then commissioned in the R.A.V.C. (S.R.). He served in France and Salonika with the 28th Division, and was mentioned in dispatches in 1918.

After being demobilised in 1919 he entered general practice in Sunderland. He was L.A.V.I. to the Sunderland Co. Borough from 1926–38, and L.V.I. to the Ministry of Agriculture Special Panel, in 1933. He was appointed Lecturer in Animal Health (Anatomy and Physiology and Veterinary Hygiene) in the Faculty of Agriculture, Durham University in 1947, and was Lecturer in Animal Health in the Durham County Council School of Agriculture from 1942–51. He has served continuously on the Council of the R.C.V.S. since 1932, and was appointed Vice-president for 1944 and President for 1945. He is a Past President of the North of England Veterinary Medical Association, and has represented that Division on Council, B.V.A.

**Dr. R. F. Montgomerie,** B.SC., F.R.C.V.S., was born in 1898, and educated at Rothesay Academy and the Royal (Dick) Veterinary College. He graduated from Edinburgh in 1923, and was awarded his B.Sc. (Edinburgh) in 1923, F.R.C.V.S. in 1925, and Ph.D. (Wales) in 1926.

He was Veterinary Investigation Officer to the University College of North Wales from 1924–37, when he was appointed Director of Veterinary Research Wellcome Foundation Limited. He was President of the N.V.M.A. from 1948–9.

**Mr. T. Stewart,** M.R.C.V.S., was born in 1918, and was educated at the Glasgow Academy and at the Glasgow Veterinary College. He qualified as a member of the Royal College of Veterinary Surgeons in 1941, and was awarded his D.V.S.M. in 1955. From 1942–3 he worked as an assistant in an agricultural practice in Wales. In 1943 he obtained a commission in the Royal Army Veterinary Corps, and was demobilised in 1946 with the honorary rank of Major.

He then became Veterinary Inspector for the Sudan Veterinary Service, a post which he held until 1949. On his return to England he was employed as Veterinary Inspector for the Ministry of Agriculture and Fisheries for seven months, and then became Senior Assistant with Messrs. Hill & Bartholemew, Ms.R.C.V.S., in their mixed practice in Darlington.

He was appointed to his present post as Lecturer in the Department of Veterinary Hygiene and Preventive Medicine of the Royal (Dick) School of Veterinary Studies of Edinburgh University in 1952.

# News and Comment

## HUNGARIAN VETERINARY SURGEONS IN THE UNITED KINGDOM

For some time past, the Association has been anxious to make contact with veterinary surgeons among the Hungarians who recently came to the U.K. after the disturbances in their own country. The first opportunity of doing so came on Friday, March 8th, when the President, Mr. Callender, entertained nine such visitors to this country at a cocktail party and informal dinner party at the Mostyn Hotel in London.

The overseas guests included one Polish and six Hungarian veterinary surgeons, as well as one second-year student, and one Hungarian whose work for his veterinary degree had been interrupted some years ago but who had, for most of his working life since then, been occupied in allied fields mostly on cattle obstetrics. The party represented a wide diversity of interests including as it did an expert on food bacteriology, two research workers (on viruses, endocrinology and the breeding and fattening of cattle), a university lecturer in pathology, and two doctors whose primary interests were horses and dogs, on the one hand, and on the other, sera and bacteriology from the industrial angle. Their personal histories over the last few years told of a courageous endurance of hardship that must command our highest admiration and respect.

In addition to Mr. Wilkinson (Senior Vice-President) and Mr. Ingram (President Elect), the B.V.A. representatives included Dr. W. R. Wooldridge, Chairman of the Parliamentary and Public Relations Committee, Miss Olga Uvarov and Mr. R. V. Blamire (both of whom have a special interest in the problems of the Hungarian refugee), and Mr. F. Knight, General Secretary of the Association. The R.C.V.S. was represented at the cocktail party only, by Professor T. J. Bosworth, Vice-President, and Mr. W. G. R. Oates, Registrar.

By common consent, the success of the evening was largely due to the efforts of Dr. Paneth, a member of the Executive of the Hungarian Relief Fund, who not only helped in some of the preliminary arrangements, but also gave valuable help as interpreter at the party, and to whom go the warmest thanks of all concerned.

At least one of the guests hoped eventually to travel on to Canada, but most of them were keen to continue their work as veterinary surgeons in this country. As one speaker put it, " We are at heart and by profession veterinary surgeons, and we want to continue that profession; we have set our hearts on it." Speakers for the Association were able to explain the legal restrictions which must inevitably delay the fulfilment of this wish, but at the same time to assure their guests that once the obstacles had been surmounted, a warm welcome awaited them as practising colleagues, and that there was indeed a place for the knowledge and experience they brought with them from Hungary.

## B.M.A. PRESS CONFERENCE

" Other professions may feel that they are involved in this," said Dr. S. Wand, Chairman of Council of the B.M.A., at a press conference held on March 20th. He was explaining to the press his Association's views on the proposed Royal Commission on Doctors' and Dentists' Remuneration, the setting up of which was announced in Parliament on February 20th.

The first paragraph of the terms of reference of the Royal Commission reads: " To consider how the levels of professional remuneration from all sources now received by doctors and dentists taking any part in the National Health Service compare with the remuneration received by members of other professions, by other members of the medical and dental professions, and by people engaged in connected occupations."

Dr. Wand made available photostat copies of a letter received in 1950 from Sir William Douglas, Permanent Secretary to the Ministry of Health. It contained the words, " The Minister agrees that the Spens report remains the basis of the remuneration of general medical practitioners."

The terms of reference of the Spens Committee were " to consider . . . what have been the normal financial expectations of general medical practice in the past, and to the desirability of maintaining in the future the proper social and economic status of general medical practice and its power to attract a suitable type of recruit to the profession."

It was explained that there is a far-reaching difference between these terms of reference. The Spens report considers the doctors' " proper social and economic status " in relation to society as a whole, and the Royal Commission considers it only in relation to members of other professions. The B.M.A. believe that the ultimate effect will be to group the professions into one self-contained class, who will not be permitted to compare their financial status with that of non-professional workers.

It is generally agreed that the professions as a whole have steadily lost ground since the war and if, from now on, they are to compare their remuneration only with that of each other and to refrain from asking for higher remuneration because their fellow professions are no better off, then Dr. Wand's statement that the terms of reference of the Royal Commission constitute a threat to every professional worker in the country would seem to be a reasonable assessment of the position.

Dr. Wand also expressed the B.M.A.'s anxiety over the ambiguity of the words " people engaged in connected occupations." The man who stokes the hospital boiler is engaged in a connected occupation; so are the mortuary attendant, the ward maid, the man who operates the hospital lift, and the cleaner who scrubs out the doctor's surgery. If any Government should adopt the principle that a professional worker's remuneration should be geared to that of unskilled workers in his own field, the possible dangers are obvious.

It could be argued that doctors and dentists within the national health scheme are in a unique position; but the Spens report could almost be speaking of our own profession when it says: " regard to length of training, to the arduousness of the General Practitioner's life compared with that in other professions, to the greater danger to health, to the skill and other qualities required, and to the degree of individual responsibility."

The B.M.A. feel that what a Royal Commission can do for one profession it can do for others, and it may be we should do well to bear that point of view in mind.

## MEMORIAL FUND FOR THE LATE MR. G. P. MALE

Friends and associates of the late Mr. G. P. Male, O.B.E., M.R.C.V.S., have formed a committee to collect subscriptions for a memorial fund. In view of his great interest in equitation it is proposed to present a championship jumping trophy to the Royal Counties Agricultural Society. Mr. Male was intimately connected with the Royal Counties Show for many years, and contributions should be sent to " The G. P. Male Memorial Fund," Lloyds Bank, Bishop's Waltham, Hampshire.

Under his will, which has just been proved, Mr. Male left £1,000 to the Victoria Veterinary Benevolent Fund.

## ISLE OF WIGHT WARBLE-FLY EXPERIMENT

An experiment was carried out in the Isle of Wight during the years 1953-6 for the purpose of determining the effectiveness of regular and adequate dressing of cattle under strict control. The Isle of Wight was chosen because it provides exceptionally favourable conditions for such an experiment, since there is comparatively little movement of cattle from the mainland to cause reinfestation and the warble-fly will not cross large stretches of water.

*Method of Operation*

During the first year of the experiment, the majority of the larger herds were inspected several times, but owing to the inadequacy of the staff it was not possible to visit every farm, and much of the information collected was obtained in discussion with farmers. In the next three years, the experiment worked much more smoothly. With the larger staff employed in 1954 and 1955, all cattle over six months were inspected each month during the dressing season; dressings were supervised and, in some cases, actually carried out by the veterinary staff. In 1956, three inspections were made of every herd that had been infested the previous year and of all herds into which imported cattle had moved since the previous dressing season; in addition, every herd that had been clear in 1955 was inspected at least once.

To ensure that everything would be ready when the veterinary officer called, a card was sent to the farmer giving him four days' notice of the intended visit. A careful record was kept of (a) the terrain of the farm; (b) the number of cattle on it; (c) degree of infestation, and (d) details of dressing.

*Results*

During the 1953 and 1954 dressing seasons, the number of warbles per infested beast varied from 10 to 40. In 1955, the average was only six. In 1956, it was rare to find a beast (apart from imported stock) with more than two warbles.

There are approximately 21,000 cattle in 600 herds on the island. The numbers of herds found to be completely free from warbles in 1954, 1955, and 1956, were 97, 374, and 500, respectively, with a corresponding reduction each year in the incidence of infestation in individual animals.

There was an annual importation of about 700 cattle from the mainland. These importations were widely distributed and constituted a serious threat to neighbouring herds that had become warble-free.

*Conclusions*

The above figures clearly show that warble-flies could be eradicated from the country by the regular and thorough dressing of infested cattle. Careful inspection and treatment are essential in the control of warble-fly. It is necessary to palpate the skin of a beast, especially the long-haired breeds; reliance on visual inspection may result in a number of warbled cattle being missed.

Brushes must be capable of holding fluid and strong enough to remove the scab. On long-haired cattle the hair over the site of the warble must be clipped away. Unless the derris penetrates the apertures, the larvae will survive.

During the past three years, the Ministry has conducted intensive publicity campaigns in which attention has been drawn to the requirements of the Warble Fly (Dressing of Cattle) Order, the serious losses of meat and milk caused by warbles, and the severe wastage of leather resulting from damaged hides.

The Warble Fly (Dressing of Cattle) Order of 1948 requires all cattle visibly infested with maggots of the warble-fly to be treated with a derris dressing *within the seven days following March 15th* or as soon afterwards as maggots appear under the skin. Until maggots cease to appear, or until June 30th, whichever is the earlier, dressings must be repeated at monthly intervals.

Research is still going on at the Ministry's veterinary laboratory in the hope of discovering a satisfactory preparation that will destroy the warbles while they are in the body of the animal. Meanwhile, progress in eradicating the warble-fly depends largely on the efforts of cattle owners themselves.

## INQUIRY INTO PRECAUTIONS AGAINST ANTHRAX

A committee of inquiry, headed by Mr. R. F. Levy, Q.C., Chairman of the Monopolies Commission, is to look into the question of precautions against anthrax. This will be the first full-scale inquiry to be carried out for 40 years and it will be chiefly concerned with protecting the health of human beings, although some aspects of the inquiry are bound also to touch on the animal health question.

In 1955, the last year for which full details are available, there were 15 cases among human beings

in this country but none was fatal. This was the lowest annual total of any year since 1946 when there were 14 cases, involving one death. In 1956 there were 1,245 cases of anthrax among animals and there were 764 cases in 1955.

The terms of reference for this committee will be to consider the existing legal provisions concerning the importation of goods infected or likely to be infected with anthrax and the precautions to be taken in connexion with such imported goods; and to make recommendations. Under the existing law, some goods likely to be infected with anthrax may not be imported at all. Others may be imported subject to disinfection at the Government Wool Disinfecting Station at Liverpool. Some can be imported subject to certain precautions to be taken at the handling factory. Others may be imported freely.

Present legislation is based on the recommendation of the 1913 Departmental Committee on anthrax. Following the passing of the Anthrax Prevention Act in 1919, the Government Wool Disinfecting Station was built at Liverpool in 1921.

## PRESERVATION OF WILD LIFE ON ST. KILDA

At a conference held on March 7th the future of wild life on the islands of St. Kilda in the Outer Hebrides was discussed. The Air Ministry at the end of 1960 expect to have radar and other installations built on the main island of Hirta and a permanent force of up to 50 civilians and military stationed there. Security regulations will not prevent free access to St. Kilda, a facility which the National Trust intends to provide if and when funds become available.

Workers and defence personnel will be prohibited from keeping pets such as dogs and cats which might cause depredations among the famous gannet and fulmar colonies, or to the St. Kilda fieldmouse. Every precaution will be taken to prevent the entry of rats, an even greater danger.

The National Trust, the Nature Conservancy, and Edinburgh University's School of Scottish Studies are all concerned that St. Kilda should be preserved intact.

This is to enable more comprehensive scientific research than has hitherto been possible to proceed into the riches of island wild life, archaeology, and social anthropology. Plans include the appointment of a permanent warden, an expedition to record archaeological features, physical geography, and agricultural, architectural, and philological aspects, and the possibility of establishing St. Kilda as a bird observatory.

## POULTRY STOCK IMPROVEMENT PLAN, 1957 REGISTER

The 1957 Register of accredited poultry breeding stations, accredited and approved hatcheries, and standard-bred breeders in England and Wales has now been published. The Register shows the breeds and approximate number of accredited and standard-bred stock on each breeding station and is published to assist poultry keepers to obtain good stock.

Copies of the Register may be obtained, post free, on application to the Poultry Branch of the Ministry of Agriculture, Fisheries and Food at Whitehall Place, London. S.W.1, or to the offices of county agricultural executive committees. Members of the Plan will receive copies direct from county poultry advisory officers. "County Lists" of accredited breeders, accredited and approved hatcheries, and standard-bred breeders, are also obtainable from county and provincial offices of the National Agricultural Advisory Service.

Purchasers of accredited, approved, or standard-bred stock may obtain *post-mortem* examinations free of charge on the carcase of any bird which dies within 28 days of leaving an accredited breeding station or an accredited or approved hatchery. Carcases sent for examination under this arrangement should be accompanied by a statement showing the name and address of the breeding station or hatchery from which the stock was bought and the date on which it was delivered. Carcases must be securely packed in a box with enough absorbent material to prevent leakage of any of the contents during transit; carriage must be pre-paid and the package should bear the name and address of the sender.

Poultry keepers in Cumberland, Westmorland, Northumberland, Durham, Lancashire, or Yorkshire should send *post-mortem* specimens to the Veterinary Laboratory, Eskgrove, Lasswade, Midlothian (Station: Bonnyrigg, Scottish Region). Specimens from the rest of England and from Wales should be sent to the Central Veterinary Laboratory, New Haw, Weybridge, Surrey (Station: Addlestone, Southern Region).

This arrangement does not relieve any person of his obligation to notify the police immediately if he suspects the existence of fowl pest.

## OVERSEAS DEMANDS FOR CLEVELAND BAY HORSES

The Director of Remount Veterinary Farms in Pakistan, Brigadier M. Z. Khan, M.R.C.V.S., has notified the Cleveland Bay Horse Society that some mares will be required as soon as the colder weather arrives. Cleveland Bay stallions have been used to up-grade local stock, and the experiment has proved very successful.

Canadian buyers are now showing considerable interest in Cleveland Bay mares, while the British Bloodstock Agency has received several requests from the Japanese Imperial Household Board. Although these horses are primarily intended for use by the Imperial household, they are ultimately to be used for breeding purposes, and both colts and fillies are required.

## ANIMAL BOARDING ESTABLISHMENTS BILL

There being a quorum present, the Committee Stage of the Animal Boarding Establishments Bill commenced on Thursday, March 7th, before Standing Committee Room C of the House of Commons, to which had been added certain other Members interested in the Bill, making a total of 36 Members of the Committee.

The first important amendment to be considered was to remove the words " for gain " from Clause 1 (1), so that instead of reading " No person shall for gain keep a boarding establishment for animals except under the authority of a licence . . ." it would read " No person shall keep a boarding establishment for animals . . ."

Several of the speakers were in agreement with this principle, and it was pointed out that it was as necessary for the boarding establishments of animal welfare societies to be inspected as any other premises. Another point made was that certain kind-hearted people maintained boarding establishments for charity, and it was as necessary to maintain proper standards in such an establishment as it was in commercial establishments.

The B.V.A. has also put forward certain amendments to this Bill. These include a request that it should be made obligatory in the local authority to have a veterinary inspection of the premises before a licence is actually issued. Also that quarantine kennels authorised by the Ministry of Agriculture, Fisheries and Food should be specifically mentioned as being exempt from the provisions of the Bill.

Amongst the speakers from the Conservative Benches were Mr. Julian Ridsdale, the Presenter of the Bill, Mr. Charles Doughty, and the Joint Under-secretary of State for the Home Department, Mr. J. E. S. Simon, and also Sir Jocelyn Lucas, who stated that he was a member of the Council of the Royal Veterinary College, and a member of the Kennel Club. For the Labour Benches, the speakers were Mr. A. Moyle and Mr. Hector Hughes.

Whilst Sir Jocelyn Lucas was speaking to the first amendment, Mr. Doughty, who is opposed to the Bill, asked the Chairman what was the quorum in the Committee, and on being informed by the Chairman that the quorum was 15, including the Chairman, Mr. Doughty left the Committee Room.

There then being less than 15 members present, after waiting for a period under standing orders, the Chairman had to announce that the Committee was adjourned again until Thursday, March 14th.

The President and the Registrar of the Royal College of Veterinary Surgeons were present throughout the proceedings.

## CORRECTION

We regret that an error was inadvertently made in our issue of March 9th (*Vet. Rec.* **69.** 333). It was reported that Mr. R. E. Glover had received an " honorary degree " of Doctor of Science from the University of London, but in fact the degree was awarded on published work. Our note was based on information received from a correspondent of standing and was published in good faith; we apologise to the University of London and to Dr. Glover for any inconvenience or embarrassment it may have caused.

## PERSONAL

Dr. Arthur Walton of the Animal Research Station, Cambridge, has been awarded the Royal Agricultural Society of England's Research Medal for 1957 for his pioneer work in the development and application of artificial insemination.

### Marriage

JOHNSON—FRANCE.—At Quorn, Leicestershire, on March 2nd, 1957, Roger Johnson, B.V.SC., M.R.C.V.S., of Castle Cary, Somerset, to Dinah France, B.V.SC., M.R.C.V.S., of Quorn, Leicestershire.

### Birth

HARRIS.—On March 6th, 1957, at Portway, Stratford sub-Castle, Salisbury, to Rosemary (*née* Street), wife of Kenneth A. Harris, M.R.C.V.S., a son, Robert William Kenneth, a brother for Fiona and Jill.

## R.C.V.S. OBITUARY

We record with regret the death of the following member of the profession:—

THOMPSON, Herbert, 19, Adelaide Road, Sheffield, 7. Graduated Edinburgh, May 24th, 1894. Died March 1st, 1957, aged 85 years.

## COMING EVENTS

### March

20th (Wed.). Meeting of the Section of Comparative Medicine, Royal Society of Medicine, at the Society's house, 5 p.m.

Ordinary General Meeting of the Sussex Veterinary Society in the Old Ship Hotel, Brighton, 2.30 p.m.

21st (Thurs.). Annual General Meeting of the East Midlands Division at the Nottingham School of Agriculture, Sutton Bonington, 2.15 p.m.

29th (Fri.). Annual Dinner-Dance of the East Midlands Division at the George Hotel, Nottingham, 7 p.m.

28th (Thurs.). Meeting of the British Veterinary Poultry Association at the Royal Veterinary College, Camden Town, N.W.1, 11 a.m.

73rd Annual General Meeting of the Western Counties Veterinary Association at the Rougemont Hotel, Exeter, 2.35 p.m.

Extraordinary General Meeting of the Central Veterinary Society at the Royal Veterinary College, Camden Town, N.W.1, 6 p.m.

30th (Sat.). Annual Convention of the Ceylon Veterinary Association in the Queen's Hotel, Kandy, Ceylon.

### April

3rd (Wed.). Meeting of the Southern Counties Veterinary Society at the Grand Hotel, Bournemouth, 7.30 p.m.

6th (Sat.). Annual Dinner of the B.R.X. Club at the Royal Veterinary College, Camden Town, N.W.1, 6 p.m.

9th (Tues.). 19th Ordinary General Meeting of the A.V.T. & R.W.s. in the Lecture Theatre No. 1, Royal Veterinary College, Camden Town, N.W.1, 11.30 a.m.

10th (Wed.). Ordinary Meeting of the Ayrshire Veterinary Association in the Station Hotel, Ayr, 2.30 p.m.

20th (Sat.). Annual Dinner of the R.V.C. '52 Club at Ye Miller of Mansfield, Goring.

Meeting of the Scottish Group of the Nutrition Society in Aberdeen for a Symposium on Clean Food.

**May**

8th (Wed.). UFAW Symposium on " Human Technique in the Laboratory " in the Harkness Hall, Birkbeck College, Malet Street, London, W.C.1, 9.45 a.m.

24th (Fri.). 165th Annual Ball of the Royal Veterinary College Students' Union Society at the Royal Veterinary College, Camden Town, N.W.1, 9 p.m.

## BRITISH VETERINARY ASSOCIATION QUARTERLY MEETINGS IN EDINBURGH

**Wednesday, April 10th—Royal (Dick) School of Veterinary Studies**

10.15 a.m. Small-Animals Committee.
2.15 p.m. Home Appointments Committee.
4.00 p.m. Farm Live-stock Committee.

**Thursday, April 11th—Royal (Dick) School of Veterinary Studies**

10.00 a.m. Veterinary State Medicine Committee.
11.30 p.m. Parliamentary and Public Relations Committee.
2.15 p.m. General Purposes and Finance Committee.

**Friday, April 12th—Royal (Dick) School of Veterinary Studies**

10.15 a.m. Council Meeting.

## ADDRESSES OF DISEASE INFECTED PREMISES

The list given below indicates, first the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak

### Anthrax

*Ayr.* Blackwood Farm, Kilmarnock (Mar. 4).
*Hereford.* Priors Grove, Putley, Ledbury (Mar. 7).
*Herts.* Sheepcote Farm, Great Amwell, Ware (Mar. 4).
*Lancs.* Halewood Farm, Gerrards Lane, Halewood, Liverpool (Mar. 5).
*Lincs.* Low Farm, North Carlton (Mar. 4).
*Norfolk.* Grange Farm, North Runcton, Kings Lynn (Mar. 5).
*Northants.* The Limes, Huntingdon Road, Thrapston, Kettering (Mar. 5).
*Stirling.* Ballmalloch Farm, Kilsyth, Glasgow (Mar. 7).
*Surrey.* Loseley Park Estate, Arlington, Guildford (Mar 5).
*Sussex* Abingworth Farm, Thakeham, Pulborough; Town House Farm, Thakeham, Pulborough (Mar. 5).
*Wilts.* Thurlston Farm, Chapmanslade, Westbury (Mar. 4).
*Yorks.* Chapel House Farm, Kilnsey, Skipton (Mar. 7).

### Fowl Pest

*Lancs.* Hillock Farm, Knowle Lane, Little Hoole; Bank View Farm, George Lane, Banks, Southport; Moorfield Farm, Moss Lane, Little Hoole (Mar. 4); Sunnyhurst, Grange Lane, Hutton; Barrons Farm, Tarleton (Mar. 5); Moss House Farm, Moss Lane, Little Hoole (Mar. 6); Belmont, High Barn Lane, New Longton; Glenarm, Liverpool Old Road, Much Hoole; Riddcar Villa, Smithy Lane, Much Hoole (Mar. 7).
*Yorks.* Springfield Poultry Farm, East Ardsley, Wakefield (Mar. 6).

### Swine Fever

*Beds.* Village Farm, Stagsden (Mar. 11).
*Berks.* Home Farm, South Hill Park, Easthampstead, Bracknell (Mar. 7).
*Bucks.* Woodend House, Medmenham, Marlow (Mar. 4); Burborough Farm, Ludgershall, Aylesbury (Mar. 6); 32, Worminghall, Aylesbury (Mar. 8); Seven Hills Farm, Seven Hills Road, Iver Heath (Mar. 11).
*Cambs.* 21, Main Street, Little Downham, Ely (Mar. 7); Field Farm, Upware Road, Wicken, Ely (Mar. 8).
*Essex.* Wallaces Farm, Halstead (Mar. 4).
*Flints.* Rhydygaled Farm, Mold (Mar. 4); Bryn Farm, Bryn-y-Baal, Mold (Mar. 8).
*Hants.* The Piggeries, Windmill Hill, Alton (Mar. 7); Barnetts Wood Farm, Brighton, Alresford (Mar. 8).
*Herts.* Down Hall Farm, Abington Pigotts, Bassingbourn, Royston (Mar. 11).
*Kent.* Portland Nurseries, Hastings Road, Marden, Tonbridge (Mar. 5); Oak Lodge Farm, Beckenham Road, West Wickham (Mar. 8).
*Midlothian.* 11, Wester Hales, Slateford, Edinburgh (Mar. 11).
*Norfolk.* Old Hall Farm, Briggate, Honing, North Walsham (Mar. 7).
*Notts.* " Fairview," Normaton Hills, East Leake, Loughborough (Mar. 6); Baulker Farm, Farnsfield, Newark; 25, Landcroft Lane, Sutton Bonington (Mar. 11).
*Oxford.* 95, High Street, Kidlington (Mar. 7).
*Surrey.* Dunce Farm, Dunsfold, Godalming (Mar. 6).
*Warwicks.* Brickhill Cottage, Heath Road, Coleshill (Mar. 7).

# CORRESPONDENCE

*The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.*

### The R.C.V.S. Council Election

Sir,—As a candidate for election to the Council of the Royal College of Veterinary Surgeons I seek the hospitality of your columns in order to state my policy if elected to Council R.C.V.S.

The Veterinary Surgeons Act of 1948 which brought about the reconstitution of the Council R.C.V.S. gave that Council the power to supervise educational standards for admission to the profession, to maintain discipline within the profession, and to prescribe an annual membership fee not exceeding £5 5s. The Act also restricted practice to members of the R.C.V.S. and members of the S.V.R. These powers are similar to those of the G.M.C. under the Medical Act.

The efficient application of these powers is of supreme importance to the dignity and even survival of the profession and it is my opinion that the interests of the profession could best be served if the Council R.C.V.S. confined its activities to the administration of the Act. Although the policies of the B.V.A. and the R.C.V.S. in representing the profession have been in general agreement up till now there is no guarantee that this will continue. Therefore, in order to avoid the possibility of a split between the two bodies with its consequent danger to the profession, the task of looking after the interests of the profession should be left to the B.V.A. whose office bearers are more representative of the profession. They are all members of the veterinary pro-